(1) $v_i = [\, x_i - (\mu_{M0} + \mu_{M+}) / 2 \,]$

wherein vi is the selective vote, xi is the expression level in the tumor sample, and μMO and μM+ are the metastatic classes of reference samples, and wherein said votes are summed in order to obtain total votes for the non-metastatic (V_{M0}) and metastatic (V_{M+}) classes; and,

(2) Prediction Strength = $[\, (V_{M0} - V_{M+}) / (V_{M0} + V_{M+}) \,]$

wherein Prediction Strength values range between 0 and 1.

In claim 21, correct the name to TLK286.

In claim 23, correct the name to SCH66336.

In claim 18, correct the spelling of metastasis

Claim 26 (new) The method of claim 18, wherein said upregulated tumor gene is the gene for PDGFRA or a gene downstream from said PDGFRA gene.

Claim 27 (new) The method of claim 26, wherein said downstream gene is selected from the group consisting of RAS, MAP2K1/MAP2K2, phosphoinositide-3-kinase, VEGF, MAP kinase, and glutathione-S-transferase.

In claim 19, please change 18 to 26.

In claim 20, please change 18 to 26.

In the Abstract:

Please replace the original Abstract with the following:

Expression profiling of primary medulloblastomas, clinically designated as either metastatic (M+) or non-metastatic (M0), identified 85 genes whose expression differed significantly between classes. A class-prediction algorithm based on these genes assigned sample class to these tumors (M+ or M0) with 72% accuracy and to four additional independent tumors with a 100% accuracy. Class prediction also assigned the metastatic medulloblastoma cell line Daoy to the metastatic class. Notably upregulated in the M+ tumors were platelet derived growth factor receptor alpha (*PDGFRA*) and members of the downstream RAS/mitogen-activated protein kinase (*MAPK*) signal transduction pathway. . These results provide the first insight into the genetic regulation of medulloblastoma metastasis and are the first to suggest a role for and the *RAS/MAPK* signaling pathway in medulloblastoma metastasis. Inhibitors of *PDGFRA* and *RAS* proteins, among others overexpressed M+ genes identified herein, represent novel therapeutic targets in